DOI:
10.1161/STROKEAHA.111.000655

Document Version
Peer reviewed version

Trends in risk factor prevalence and management prior to first stroke: data from the South London Stroke Register 1995–2011

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Authors' final draft; final published version available at:
http://stroke.ahajournals.org/content/44/7/1809.full.pdf?ijkey=Tysogij5tRimuoB

Ethics

The study was approved by the ethics committees of Guy's and St Thomas' Hospital Trust, King's College Hospital, Queen's Square, and Westminster Hospital.

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4498 words

Abstract

Background and purpose Vascular risk factors are suboptimally managed internationally. This study investigated time trends in risk factors diagnosed prior-to-stroke and their treatment, and factors associated with appropriate medication use.

Methods 4416 patients with a first stroke were registered in the population-based South London Stroke Register from 1995–2011. Previously diagnosed risk factors and usual medications were collected from patients' primary care and hospital records. Trends and associations were assessed using multivariate logistic regression.

Results 72% of patients were diagnosed previously with one or more risk factor; 30% had diagnosed risk factors which were untreated. Hypercholesterolemia increased significantly over the study period; MI and TIA prevalences decreased. Antiplatelet prescription increased in AF, MI, and TIA (AF: 37%–51%, P<0.001; MI: 48%–69%, P<0.001; TIA: 49%–61%, P=0.015). Anticoagulant prescription for AF showed a non-significant increase (12%–23%; P=0.059). Fewer older patients with AF were prescribed anticoagulants (age > 85 v < 65: aRR 0.19, 95% CI 0.08–0.41). Black ethnicity (aRR 1.17, 95% CI 1.10–1.23) and female sex (aRR 1.09, 95% CI 1.03–1.15) were associated with increased antihypertensive drug prescription; other medications did not vary by ethnicity or sex.

Conclusions Antiplatelet and cholesterol-lowering treatment prescribing have improved significantly over time; however, only a minority with AF received anticoagulants, and this did not improve significantly. Overall, 30% of strokes occurred in patients with previously diagnosed but untreated risk factors.

Background

Stroke remains a major preventable cause of morbidity and mortality internationally.[1] In addition to lifestyle modification, cost-effective drug treatments for hypertension, hypercholesterolemia, and atrial fibrillation (AF) reduce stroke risk, heart disease and mortality.[2–4] Despite international guidance aimed at improving primary prevention, risk factor control rates remain low.[5–7] Suboptimal control is associated with inadequate risk factor detection and treatment, ethnic differences in risk factor susceptibility and response to treatment, socio-economic deprivation, and poor treatment adherence.[5,7–9]

Several studies have found ethnic differences in stroke risk factors. A US case-control study found that hypertension and diabetes were significantly more prevalent among black than white stroke patients, whereas AF was significantly more prevalent in white patients.[10] Similar results were reported by the South London Stroke Register (SLSR) from 1995–98.[11] A US cross-sectional study found that apparent ethnic differences in stroke risk factors were explained by differences in income.[12]

We sought to examine trends from 1995–2011 in prior-to-stroke risk factors and use of appropriate treatment, using data from the SLSR. We aimed to investigate variation in risk factors by age, sex, ethnicity, socio-economic group, and stroke subtype, and factors associated with appropriate treatment.

Methods

The methods of the SLSR have been described previously,[13] and are summarised below. The SLSR is a population-based register recording all first strokes in a defined region of Lambeth and Southwark, with a population of 310,028 according to the 2001 UK Census, with 63% white, 28% black (9% black Caribbean, 15% black African, and 4% black other), and 9% other ethnic group. By 2011, the source population had increased to 357,308, with 56% white, 25%

black (7% black Caribbean, 14% black African, 4% black other) and 18% other. The largest increase was in those aged 49–59 (49%); the proportion aged 65+ fell by 10%.

Overlapping notification sources were used to increase data completeness. Data were collected by study nurses and field workers. Stroke diagnosis was confirmed by a study clinician according to WHO criteria. Ethnicity was self-reported using 1991 UK census criteria. To increase numbers per group, white British and white other were considered as white ethnicity; black Caribbean, black African, and black other were grouped as black ethnicity. Risk factors recorded prior-to-stroke (hypercholesterolemia [from 2001], hypertension, AF, myocardial infarction [MI], transient ischemic attack [TIA], and diabetes) and usual prescribed medication (antiplatelets, anticoagulants, antihypertensive drugs, and cholesterol-lowering drugs) were collected from patients' general practitioners and hospital records.

Stroke subtype was determined from CT or MRI results where available and classified as ischemic, intracerebral hemorrhage (ICH), subarachnoid hemorrhage (SAH), or undefined. Deprivation was estimated using the Carstairs index, which combines male unemployment, overcrowding, car ownership and proportion in social classes IV and V in a small area.[14] The index was derived from 2001 census data for each lower layer super output area (SOA), covering an average population of 1500. Scores were obtained from patients' home postcodes at the time of stroke.

Data were analysed in four-year groups to increase numbers per group. Demographic trends were assessed using the Chi-squared test for trends. Risk factor and medication trends were assessed in logistic regression models adjusting for age, sex, ethnicity, stroke subtype, and deprivation with the year of stroke as an explanatory variable. Associations with risk factors and prescribed medication were assessed in logistic regression models incorporating sex, age, ethnicity, stroke subtype, deprivation, and year of stroke. P values <0.05 were regarded as statistically significant. Risk ratios were estimated using Zhang and

Yu's method.[15] Inter-variable interactions were assessed for each analysis. Analyses omitted patients with missing data. Analyses were conducted using R.[16]

Results

Between January 1995 and 2011, 4416 patients were registered (table 1). Patient median age was 72.4 years (interquartile range 61.2–81.1); ethnicities were white (70.5%), black (21.3%; 13.0% black Caribbean, 7.6% black African, and 0.6% black other), and other (5.7%). Stroke subtypes were ischemic (73.8%), ICH (12.7%), SAH (5%), and undefined (8.4%). White and black patients had significantly lower Carstairs scores than other ethnicities, but the difference was small (mean score [higher=more deprived]: white 9.421, black 9.662, other 10.21; P=0.006). Data completeness was high for all variables (ethnicity 97%, stroke subtype 96%, risk factors 95–97%; prescribed medication 96–97%). There were no significant inter-variable interactions in any analysis.

Risk factor trends

Risk factor trends are reported in figure 1 and table 1. 72% of patients had one or more risk factors diagnosed prior-to-stroke. Overall risk factor prevalences were: hypertension, 64%; hypercholesterolemia, 24%; AF, 16%; diabetes, 19%; prior MI, 11%; and prior TIA 12%. Hypercholesterolemia significantly increased over time (10.5%–31.7%, P<0.001); prior-to-stroke MI and TIA significantly reduced (MI 7.7%–2.7%, P<0.001; TIA, 16.3%–8.9%, P<0.001). Hypertension, AF, and diabetes did not change significantly over time.

Risk factor associations

The multivariate analyses are reported in table 2. Hypertension, diabetes, AF, prior MI and TIA increased significantly with age. Hypercholesterolemia was highest in those aged 65–74. Hypertension and MI were significantly more prevalent in men; other risk factors were not significantly different between men and women.

Black patients had significantly greater prevalences of hypertension and diabetes than white patients (adjusted RRs [aRR]: hypertension 1.22, 95% CI 1.17–1.27; diabetes 2.15, 95% CI 1.91–2.39) and significantly lower AF, MI, and TIA (AF: 0.47, 95% CI 0.35–0.60; MI: 0.58, 95% CI 0.43–0.77; TIA: 0.76, 95% CI 0.59–0.96). There was no association between deprivation and any risk factor. Hypertension prevalence was similar in ischemic stroke and ICH, but significantly lower in SAH (aRR *v* ischemic stroke 0.63, 95% CI 0.52–0.75). Hypercholesterolemia, diabetes, prior MI, and AF were significantly less prevalent in ICH and SAH than ischemic stroke.

Prescribing trends

Trends in prescribed medication are reported in table 1 and figure 2. 26% of patients had a single untreated risk factor, 13% had 2 or more. The proportions of those with risk factors prescribed appropriate treatment were: hypertension 62%; hypercholesterolemia 75%; MI (antiplatelets) 62%; AF 64% (anticoagulants 17%; antiplatelets 48%; [1% both]); TIA (antiplatelets) 58%. Prescribed treatment for hypercholesterolemia increased over time (70%–77%, P=0.004). Antiplatelet prescription for AF significantly increased (37%–51%, P<0.001); anticoagulant prescription increased, but was not significant (12%–23%, P=0.059). Antiplatelet prescription in MI and TIA significantly increased over time (MI, 48%–60%, P<0.001; TIA, 49%–61%, P=0.015). Antihypertensive prescription did not significantly change over time.

Associations with appropriate treatment

The multivariate analyses are reported in table 3. Anticoagulant prescription in AF for older patients was low, and least in those aged ≥85 (aRRs *v* <65s; 65–74: 0.41, 75–84: 0.77, ≥85: 0.19). Significantly more women with hypertension were treated than men; there were no significant differences for other risk factor treatments between sexes.

Significantly more black patients with hypertension were treated than white patients (aRR 1.17, 95% CI 1.10–1.15). There was no significant association between other risk factor treatments and ethnicity, or between deprivation and

any risk factor treatment. ICH and SAH were associated with significantly higher anticoagulant prescription (aRR *v* ischemic stroke: ICH 3.14, 95% CI 2.21–4.04; SAH 4.64, 95% CI 2.40–5.72).

Discussion

This paper analyses trends in the prevalence and treatment of risk factors prior-to-stroke over 15 years. Hypercholesterolemia increased significantly over time, and prior MI and TIA fell. Prescribing of antiplatelets and cholesterol-lowering treatments significantly increased over the study period. A minority with AF were prescribed anticoagulants; this did not significantly improve over time, and was least likely in older people. Overall, one third of first strokes occurred in people who were not prescribed treatment for a previously diagnosed risk factor.

Anticoagulants are effective for the prevention of AF-related stroke; a 2007 meta-analysis found anticoagulation was substantially more effective than aspirin.[17] A UK consensus statement published after the SLSR data were collected recommended that aspirin is no longer used for stroke prevention in AF.[18] Anticoagulant prescribing for AF remained low throughout the study period, and was lowest in older patients, among whom AF was most prevalent. UK research found significantly lower primary prevention use among older people, despite advancing age being the most important risk factor for vascular disease.[19] The SLSR did not record contraindications to anticoagulants, though US research found low warfarin use even among those with no contraindications.[6]

These results provide an example of delay in implementing evidence-based practice.[20] Guidelines recommending anticoagulation for AF were published in the early 1990s.[21] Barriers to anticoagulant use have been examined in qualitative research, and include perceived high rates of bleeding, particularly in the elderly, and clinicians' perceptions that patients would not agree to treatment.[22] However, an RCT in people aged over 75 found that warfarin was more effective than aspirin in preventing stroke, with no increase in

hemorrhage.[23] The SLSR data suggest that there is room for improvement in the use of anticoagulation in AF.

Consistent with previous studies,[10] this study found that AF was substantially less prevalent in black than white stroke patients. This difference was not caused by underdiagnosis: a similar discrepancy was found on ECG on hospital admission. This difference may be explained by lower AF prevalence in black people in the general population,[24] and ethnic differences in the etiological role of AF in stroke.[25]

RCTs of anticoagulation in AF were conducted overwhelmingly in white populations; only 6% of participants were non-white.[25] Additionally, tools for identifying AF patients at highest risk, $CHADS_2$ and CHA_2DS_2-VASc, do not incorporate ethnicity and have not been validated in black populations.[26,27] A US observational study found increased warfarin-related intracranial hemorrhage in black compared with white patients.[28] These data question whether the balance of benefit and harm with anticoagulation may vary among different ethnic groups.[28]

Previous studies have found that ethnic minority populations have inadequate access to healthcare.[29] We found no difference in risk factor treatment between ethnic groups, except for hypertension, with significantly more black patients treated than white patients. Here, we did not assess risk factor control, but merely whether treatment was prescribed. UK observational studies have reported significantly worse control in black compared with white patients.[30, 31]

There was a reduction in patients with untreated risk factors from 40% in 1995-98 to 17% in 1999-2002, followed by increases until 2010, although the overall trend was not significant. This change from 1995-98 to 1999-2002 was principally caused by a large increase in hypertension treatment from 52-74%. This improvement was not sustained; rates reduced to 55% by 2007-10. The reason for these changes is unclear, but migration in the source population may

have contributed. According to census data, from 1991–2001 the number of black African residents increased (7-15%) and the number of white fell (72-63%).

In the SLSR, deprivation was not associated with significant differences in risk factors or prescribed treatments, and was not responsible for ethnic differences in risk factors. This contrasts with US research, which reported that income differences explained much of the difference in risk factor prevalence between white and African American patients.[12] This discrepancy may reflect differences in healthcare for deprived populations between the UK and US; US research has found inadequate risk factor management was more likely in people without health insurance.[32] The reduction in prior MI among first-stroke patients is likely to reflect a reduction in MI in the general population; possible contributing factors include increasing use of primary prevention and a 2007 UK smoking ban in public places.[33]

Risk factor prevalences are susceptible to changes in diagnostic cutoffs over time. UK hypertension guidelines have recommended similar cutoffs over the study period (>160/100mmHg, or >140/90 with other risk factors);[34, 35] diagnostic criteria for diabetes were lowered in 1999 from a fasting blood glucose ≥7.8mmol/l to ≥7.0mmol/l.[36] European guidelines on hypercholesterolemia were published in 1998, recommending a diagnostic cutoff of total cholesterol ≥5, or LDL cholesterol ≥3;[37] subsequent revisions recommend treatment based on overall cardiovascular risk.[38] The large increase in hypercholesterolemia is likely to be explained by increased detection.

Strengths and limitations

This study was population based, with multiple notification sources including hospitalised and community stroke patients. Risk factor diagnoses were collected from patient medical records. Though bias may occur through changes in documentation practice over the time period, this was mitigated by using both hospital and GP records. This study did not collect individual blood pressure or serum cholesterol values; therefore, the results represent rates of detected risk factors and omit those who were unaware.

It is not possible to draw conclusions from this study about primary prevention uptake in the general population. The stroke population is more likely to contain people with multiple and inadequately treated risk factors. Indeed, the number of people with a first stroke reduced over time, which could be consistent with improvements in prevention. This study does provide evidence, however, that a substantial number of stroke patients were not prescribed optimal treatment for previously diagnosed risk factors.

Conclusions

There have been significant improvements in the use of appropriate antiplatelet and cholesterol-lowering treatments; however, almost one third of strokes occurred in patients with diagnosed but untreated risk factors. Anticoagulant prescription in AF remained low throughout the study period, and was lowest in older people. These results highlight a need for continued research into interventions to improve uptake of primary prevention.

Funding

This paper presents independent research commissioned by the NIHR under its Programme Grants for Applied Research funding scheme (RP-PG-0407-10184). The views expressed in this paper are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health. The SLSR has also received funding from the Northern & Yorkshire NHS R&D Programme in Cardiovascular Disease and Stroke, Guy's and St Thomas' Hospitals Charitable Foundation, the Stanley Thomas Johnson Foundation, the Stroke Association, and DH HQIP.

Conflicts of interest

None declared

		1995–1998 (n=1305)	1999–2002 (n=1074)	2003–2006 (n=994)	2007–2010 (n=877)	P (trend)
Sex	male	638 (48.9%)	535 (49.8%)	537 (54%)	429 (48.9%)	0.399
	female	667 (51.1%)	539 (50.2%)	457 (46%)	448 (51.1%)	0.399
Ethnicity	white	1028 (78.8%)	753 (70.1%)	671 (67.5%)	576 (65.7%)	<0.001
	black	216 (16.6%)	209 (19.5%)	221 (22.2%)	225 (25.7%)	<0.001
	other	52 (4%)	62 (5.8%)	73 (7.3%)	57 (6.5%)	0.002
	unknown	9 (0.7%)	50 (4.7%)	29 (2.9%)	19 (2.2%)	0.058
Age	<65	343 (26.3%)	362 (33.8%)	334 (33.6%)	303 (34.5%)	<0.001
	65–74	365 (28%)	276 (25.7%)	250 (25.2%)	189 (21.6%)	0.001
	75–84	398 (30.5%)	274 (25.6%)	283 (28.5%)	242 (27.6%)	0.247
	≥85	198 (15.2%)	160 (14.9%)	127 (12.8%)	143 (16.3%)	0.954
Subtype	ischemic	916 (70.2%)	786 (73.2%)	776 (78.1%)	699 (79.7%)	<0.001
	ICH	177 (13.6%)	163 (15.2%)	124 (12.5%)	88 (10%)	0.009
	SAH	71 (5.4%)	71 (6.6%)	51 (5.1%)	20 (2.3%)	0.001
	undefined	141 (10.8%)	54 (5%)	43 (4.3%)	70 (8%)	0.001
Risk factors	hypertension	845 (69.2%)	555 (56.8%)	630 (65.1%)	545 (63.4%)	0.091
	hypercholesterolemia	–	97 (10.5%)	226 (23.7%)	272 (31.7%)	<0.001
	diabetes	209 (17%)	178 (18%)	194 (20.3%)	180 (20.7%)	0.166
	AF	252 (20.6%)	138 (13.9%)	148 (15.3%)	127 (14.9%)	0.13
	prior MI	90 (7.7%)	37 (4%)	48 (5%)	23 (2.7%)	<0.001
	prior TIA	196 (16.3%)	105 (10.6%)	111 (11.5%)	76 (8.9%)	<0.001
Treatments	≥1 untreated risk factor	524 (40.2%)	184 (17.1%)	236 (23.7%)	311 (35.5%)	0.513
	treated hypertension	433/827 (52.4%)	373/507 (73.6%)	460/618 (74.4%)	297/540 (55%)	0.502
	treated hypercholesterolemia	–	54/81 (66.7%)	175/223 (78.5%)	210/270 (77.8%)	0.001
	AF (antiplatelet)	86/232 (37.1%)	52/101 (51.5%)	84/147 (57.1%)	64/125 (51.2%)	<0.001
	AF (anticoagulant)	30/245 (12.2%)	21/124 (16.9%)	30/147 (20.4%)	29/125 (23.2%)	0.059
	MI (antiplatelet)	75/155 (48.4%)	55/77 (71.4%)	74/96 (77.1%)	45/75 (60%)	<0.001
	TIA (antiplatelet)	86/174 (49.4%)	56/82 (68.3%)	71/111 (64%)	46/75 (61.3%)	0.015
	TIA (anticoagulant)	6/192 (3.1%)	5/83 (6%)	4/111 (3.6%)	5/75 (6.7%)	0.349

Table 1– Trends in demographics, prior-to-stroke risk factors, and treatments

	hypertension	hypercholesterolemia	diabetes	AF	prior MI	prior TIA
<65*	–	–	–	–	–	–
65–74	1.16 (1.1–1.21)	1.39 (1.17–1.63)	1.55 (1.33–1.79)	1.59 (1.27–1.96)	1.7 (1.34–2.15)	1.21 (0.96–1.52)
75–84	1.19 (1.14–1.24)	1.11 (0.92–1.33)	1.23 (1.03–1.45)	2.35 (1.98–2.75)	1.69 (1.33–2.13)	1.16 (0.92–1.45)
≥85	1.16 (1.1–1.23)	0.78 (0.59–1.01)	0.85 (0.65–1.09)	3.04 (2.6–3.49)	1.76 (1.32–2.3)	1.21 (0.91–1.58)
male*	–	–	–	–	–	–
female	1.05 (1–1.09)	0.99 (0.85–1.14)	0.95 (0.83–1.08)	1.1 (0.95–1.27)	0.66 (0.54–0.8)	1.07 (0.9–1.27)
white*	–	–	–	–	–	–
black	1.22 (1.17–1.27)	0.85 (0.7–1.01)	2.15 (1.91–2.39)	0.47 (0.35–0.6)	0.58 (0.43–0.77)	0.76 (0.59–0.96)
other	1.06 (0.96–1.15)	1.06 (0.78–1.38)	2.35 (1.96–2.74)	0.39 (0.22–0.63)	0.77 (0.48–1.15)	0.86 (0.56–1.25)
unknown	0.89 (0.72–1.05)	0.7 (0.37–1.14)	1.75 (1.2–2.36)	1.13 (0.68–1.71)	1.46 (0.84–2.3)	0.71 (0.31–1.33)
ischemic*	–	–	–	–	–	–
ICH	0.95 (0.88–1.02)	0.51 (0.36–0.68)	0.55 (0.43–0.7)	0.65 (0.49–0.85)	0.58 (0.4–0.8)	0.58 (0.41–0.78)
SAH	0.63 (0.52–0.75)	0.27 (0.12–0.52)	0.23 (0.12–0.41)	0.33 (0.14–0.63)	0.45 (0.2–0.83)	0.08 (0.01–0.24)
undefined	0.96 (0.87–1.04)	0.95 (0.72–1.21)	1.12 (0.89–1.38)	0.91 (0.7–1.17)	1.23 (0.91–1.61)	0.74 (0.51–1.02)
5 years advance in time	0.97 (0.95–1)	1.51 (1.36–1.66)	1.04 (0.97–1.11)	0.93 (0.86–1)	0.87 (0.79–0.96)	0.76 (0.69–0.84)
Carstairs score (as ordinal)	1.01 (1–1.01)	1 (0.98–1.02)	0.99 (0.97–1.01)	0.99 (0.97–1.01)	1.03 (1–1.05)	1.02 (1–1.04)

Table 2 – Association of demographics and stroke subtype with risk factors: RRs with 95% CI; columns show results from a logistic regression model with the risk factor as the dependent variable; *=reference category

	hypertension	hypercholesterolemia	MI (antiplatelet)	AF (anticoagulant)	AF (antiplatelet)	TIA (antiplatelet)
<65*	–	–	–	–	–	–
65–74	1.1 (1.02–1.17)	1.12 (1.03–1.19)	1.04 (0.79–1.24)	0.41 (0.2–0.82)	1.12 (0.81–1.42)	1.29 (1.09–1.45)
75–84	1.05 (0.97–1.13)	1.18 (1.1–1.24)	1.05 (0.81–1.25)	0.77 (0.44–1.3)	1.16 (0.87–1.43)	1.18 (0.97–1.36)
≥85	0.95 (0.84–1.05)	1.21 (1.1–1.28)	0.89 (0.6–1.16)	0.19 (0.08–0.41)	1.26 (0.96–1.53)	1.28 (1.04–1.46)
male*	–	–	–	–	–	–
female	1.09 (1.03–1.15)	0.89 (0.76–0.99)	0.88 (0.71–1.05)	1.35 (0.93–1.91)	0.92 (0.75–1.1)	0.94 (0.76–1.11)
white*	–	–	–	–	–	–
black	1.17 (1.1–1.23)	0.93 (0.8–1.05)	0.92 (0.66–1.16)	0.8 (0.37–1.49)	0.94 (0.65–1.25)	0.87 (0.64–1.11)
other	1.06 (0.92–1.18)	1 (0.8–1.15)	0.7 (0.35–1.11)	1.17 (0.29–2.77)	0.87 (0.37–1.46)	1.06 (0.66–1.4)
unknown	1 (0.76–1.22)	not estimable	1.07 (0.6–1.43)	1.5 (0.47–3.11)	0.97 (0.48–1.48)	0.39 (0.07–1.01)
ischemic*	–	–	–	–	–	–
ICH	0.79 (0.69–0.89)	1.03 (0.82–1.18)	0.68 (0.4–0.99)	3.14 (2.21–4.04)	0.45 (0.24–0.74)	1.12 (0.8–1.38)
SAH	0.74 (0.55–0.93)	0.85 (0.37–1.21)	0.45 (0.08–1.11)	4.64 (2.4–5.72)	0.3 (0.02–1.06)	not estimable
undefined	0.83 (0.72–0.95)	1.08 (0.91–1.21)	1.04 (0.78–1.26)	0.71 (0.28–1.41)	1.19 (0.9–1.46)	1.14 (0.8–1.42)
5 years advance in time	1.01 (0.98–1.04)	1.09 (1.03–1.15)	1.15 (1.07–1.22)	1.21 (1.01–1.45)	1.18 (1.09–1.26)	1.11 (1.03–1.19)
Carstairs score (as ordinal)	1 (0.99–1.01)	1 (0.99–1.02)	1 (0.98–1.02)	0.98 (0.94–1.03)	1 (0.98–1.02)	0.99 (0.97–1.01)

Table 3 – Association of demographics and stroke subtype with prescribed preventative medication: RRs with 95% CI; columns show results from a logistic regression model with the risk factor as the dependent variable; *=reference category

References

1. WHO. Cardiovascular diseases (CVDs) factsheet. WHO website. http://www.who.int/mediacentre/factsheets/fs317/en/index.html. Accessed January 7, 2013

2. Wright JM, Musini VM. First-line drugs for hypertension. Cochrane Database of Systematic Reviews. 2009;CD001841.

3. Cholesterol Treatment Trialists' Collaborators. The effects of lowering LDL cholesterol with statin therapy in people at low risk of vascular disease: meta-analysis of individual data from 27 randomised trials. Lancet. 2012;380:581-90.

4. Aguilar MI, Hart R. Oral anticoagulants for preventing stroke in patients with non-valvular atrial fibrillation and no previous history of stroke or transient ischemic attacks. Cochrane Database of Systematic Reviews. 2009;CD001927.

5. Kearney PM, Whelton M, Reynolds K, Whelton PK, He J. Worldwide prevalence of hypertension: a systematic review. Journal of hypertension. 2004;22:11.

6. Go AS. Anticoagulation Therapy for Stroke Prevention in Atrial Fibrillation: How Well Do Randomized Trials Translate Into Clinical Practice? JAMA. 2003;290:2685–92.

7. Cruz-Flores S, Rabinstein A, Biller J, Elkind MSV, Griffith P, Gorelick PB, et al. Racial-Ethnic Disparities in Stroke Care: The American Experience. Stroke. 2011;42:2091–116.

8. Pears E, Hannaford PC, Taylor MW. Gender, age and deprivation differences in the primary care management of hypertension in Scotland: a cross-sectional database study. Family Practice. 2003;20:22–31.

9. WHO. Adherence to long-term therapies: evidence for action. WHO website. http://www.who.int/chp/knowledge/publications/adherence_report/en/index.html. Accessed January 7, 2013

10. Sacco RL, Boden-Albala B, Abel G, Lin I-F, Elkind M, Hauser WA, et al. Race-Ethnic Disparities in the Impact of Stroke Risk Factors: The Northern Manhattan Stroke Study. Stroke. 2001;32:1725–31.

11. Hajat C, Dundas R, Stewart JA, Lawrence E, Rudd AG, Howard R, Wolfe CDA. Cerebrovascular Risk Factors and Stroke Subtypes: Differences Between Ethnic Groups. Stroke. 2001;32:37–42.

12. Bravata DM, Wells CK, Gulanski B, Kernan WN, Brass LM, Long J, Concato J. Racial Disparities in Stroke Risk Factors The Impact of Socioeconomic Status. Stroke. 2005;36:1507–11.

13. Stewart JA, Dundas R, Howard RS, Rudd AG, Wolfe CDA. Ethnic differences in incidence of stroke: prospective study with stroke register. BMJ. 1999;318:967–71.

14. Morgan O, Baker A. Measuring deprivation in England and Wales using 2001 Carstairs scores. Health Statistics Quarterly. 2006;31:28–33.

15. Zhang J, Yu KF. What's the Relative Risk?: A Method of Correcting the Odds Ratio in Cohort Studies of Common Outcomes. JAMA.1998;280:1690–1.

16. R Core Development Team. R, A Language and Environment for Statistical Computing. Vienna, Austria; 2008.

17. Lip GY, Lim HS. Atrial fibrillation and stroke prevention. Lancet Neurology. 2007;6:981–93.

18. Stott DJ, Dewar RI, Garratt CJ, Griffith KE, Harding NJ, James MA, et al. Approaching the comprehensive management of Atrial Fibrillation: Evolution or revolution?. Royal College of Physicians of Edinburgh website. http://www.rcpe.ac.uk/clinical-standards/standards/rcpe-af-consensus-statement-2012.pdf Accessed January 7, 2013.

19. Sheppard JP, Singh S, Fletcher K, McManus RJ, Mant J. Impact of age and sex on primary preventive treatment for cardiovascular disease in the West Midlands, UK. BMJ. 2012;345:4535.

20. Haynes B, Haines A. Barriers and bridges to evidence based clinical practice. BMJ. 1998;317:273–6.

21. American College of Physicians. Guidelines for Medical Treatment for Stroke Prevention. Annals of Internal Medicine. 1994;121:54–5.

22. Bungard TJ, Ghali WA, Teo KK, McAlister FA, Tsuyuki RT. Why do patients with atrial fibrillation not receive warfarin? Archives of Internal Medicine. 2000;160:41–6.

23. Mant J, Hobbs FDR, Fletcher K, Roalfe A, Fitzmaurice D, Lip GYH, Murray E. Warfarin versus aspirin for stroke prevention in an elderly community population with atrial fibrillation (the Birmingham Atrial Fibrillation Treatment of the Aged Study). Lancet. 2007;370:493–503.

24. Alonso A, Agarwal SK, Soliman EZ, Ambrose M, Chamberlain AM, Prineas RJ, Folsom AR. Incidence of atrial fibrillation in whites and African-Americans: The Atherosclerosis Risk in Communities study. American Heart Journal. 2009;158:111–7.

25. Shen AYJ, Chen W, Yao JF, Brar SS, Wang X, Go AS. Effect of race-ethnicity on the efficacy of warfarin: potential implications for prevention of stroke in patients with atrial fibrillation. CNS drugs. 2008;22:815–25.

26. Olesen JB, Lip GYH, Hansen ML, Hansen PR, Tolstrup JS, Lindhardsen J, et al. Validation of risk stratification schemes for predicting stroke and thromboembolism in patients with atrial fibrillation. BMJ. 2011;342.

27. Gage BF, Walraven C van, Pearce L, Hart RG, Koudstaal PJ, Boode B, et al. Selecting Patients With Atrial Fibrillation for Anticoagulation Stroke Risk Stratification in Patients Taking Aspirin. Circulation. 2004;110:2287–92.

28. Shen AY-J, Yao JF, Brar SS, Jorgensen MB, Chen W. Racial-Ethnic Differences in the Risk of Intracranial Hemorrhage Among Patients With Atrial Fibrillation. Journal of the American College of Cardiology. 2007;50:309–15.

29. Szczepura A. Access to health care for ethnic minority populations. Postgraduate Medical Journal. 2005;81:141–7.

30. Lane DA, Lip GY. Ethnic Differences in Hypertension and Blood Pressure Control in the UK. QJM. 2001;94:391–6.

31. Hull S, Dreyer G, Badrick E, Chesser A, Yaqoob MM. The relationship of ethnicity to the prevalence and management of hypertension and associated chronic kidney disease. BMC Nephrology. 2011;12:41.

32. Brooks EL, Preis SR, Hwang S-J, Murabito JM, Benjamin EJ, Kelly-Hayes M, Sorlie P, Levy D. Health Insurance and Cardiovascular Disease Risk Factors. American Journal of Medicine. 2010;123:741–7.

33. Sims M, Maxwell R, Bauld L, Gilmore A. Short Term Impact of Smoke-free Legislation in England: Retrospective Analysis of Hospital Admissions for Myocardial Infarction. BMJ. 2008;340:c2161

34. Sever P, Beevers G, Bulpitt C, Lever A, Ramsay L, Reid J, Swales J. Management guidelines in essential hypertension: report of the second working party of the British Hypertension Society. BMJ. 1993;306:983–7.

35. Krause T, Lovibond K, Caulfield M, McCormack T, Williams B, et al. Management of hypertension: summary of NICE guidance. BMJ. 2011;343:4891.

36. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. Diabetic medicine. 1998;15:539–53.

37. Wood D, De Backer G, Faergeman O, Graham I, Mancia G, Pyörälä K. Prevention of coronary heart disease in clinical practice: recommendations of the Second Joint Task Force of European and other Societies on Coronary Prevention. Atherosclerosis. 1998;140:199–270.

38. JBS 2: Joint British Societies' guidelines on prevention of cardiovascular disease in clinical practice. Heart. 2005;91 Suppl 5:1.

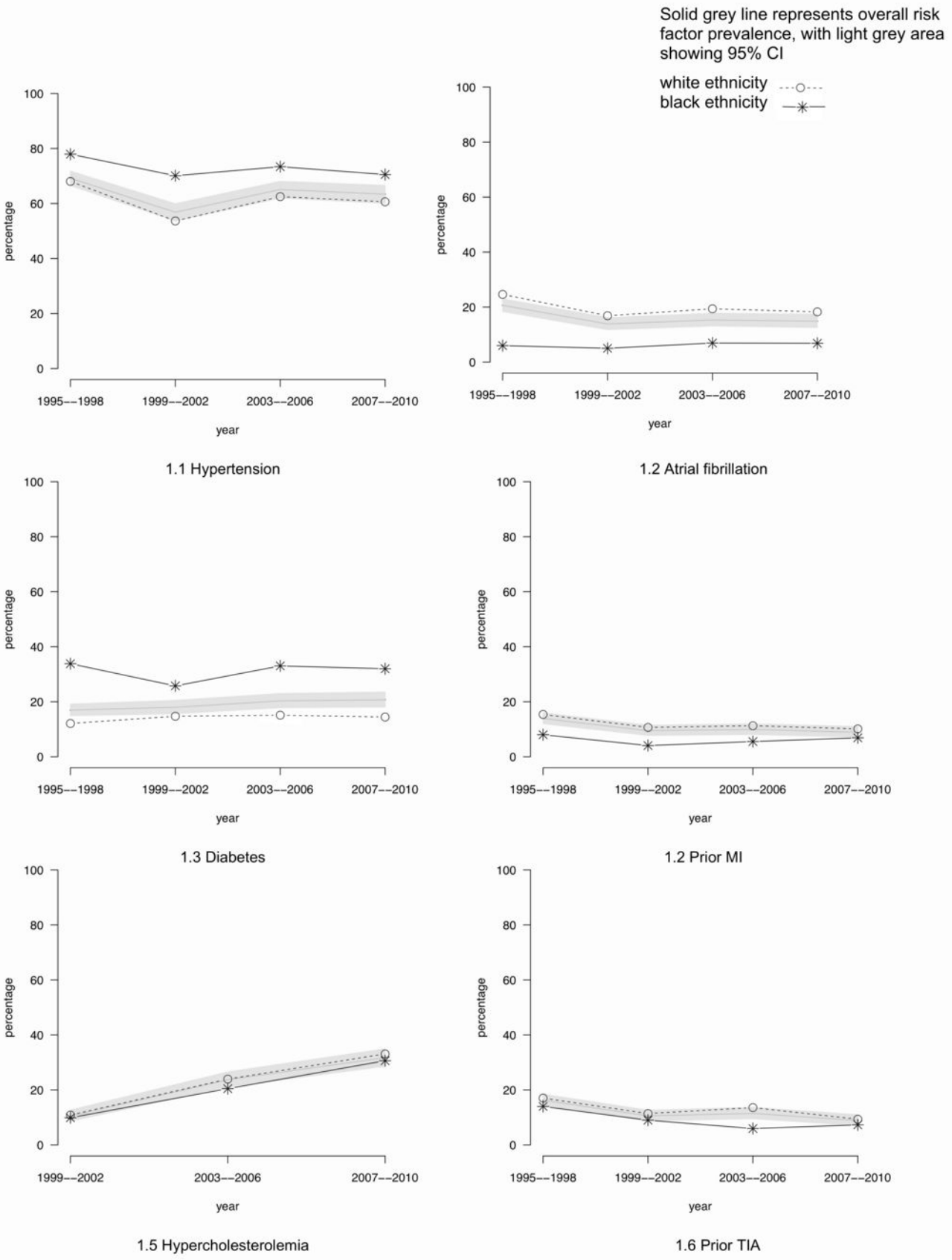

Figure 1: Trends in risk factors diagnosed prior to stroke prevalences with 95% CI, and variation by ethnic group

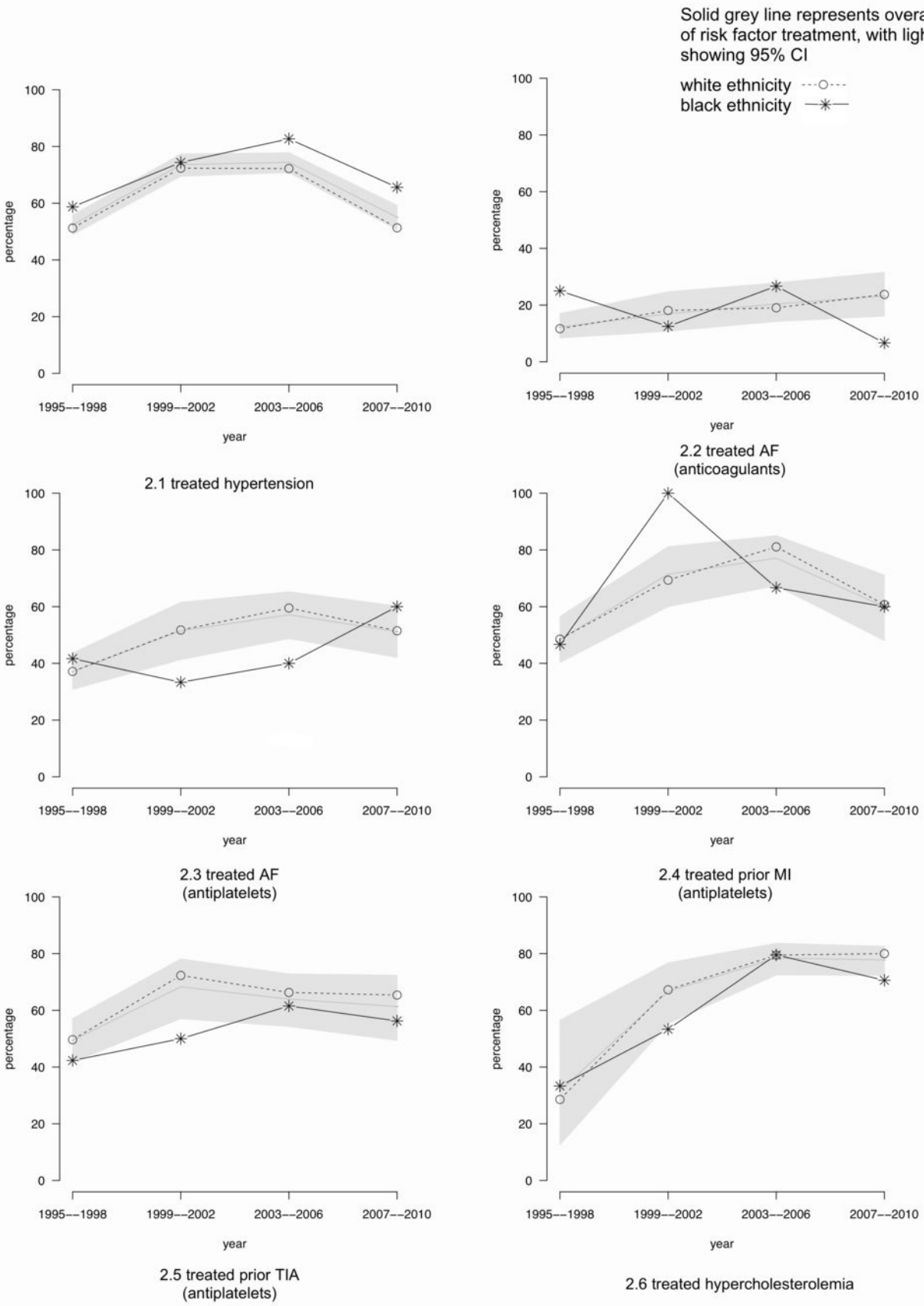

Figure 2: Trends in prescribed medication in patients with previously diagnosed risk factors: rates for all patients with 95% CIs, and variation by ethnic group